CASE REPORTS

Gastrojejunal Mucosal Prolapse After Subtotal Gastrectomy

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ALTHOUGH GASTROJEJUNAL mucosal prolapse is an infrequent complication of subtotal gastrectomy, when it does occur it may cause distressing symptoms. Since anastomotic revision may relieve these symptoms, recognition of this prolapse is important. Kirklin,[1] in 1935, mentioned such prolapse and a fairly large series was reported in 1963 by LeVine et al.[2] That the diagnostic features are still not appreciated was brought to our attention at a refresher course when a specimen case was diagnosed correctly by only a few of those attending.

Within the last year we have observed two cases in which patients with gastrojejunal mucosal prolapse benefited from surgical revision. Classic roentgenographic features were demonstrated in both, and in one we were able to observe the development of the prolapse.

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Reports of Cases

Case 1. A 48-year-old man had subtotal gastrectomy and antecolic gastrojejunostomy of Hofmeister type in 1954. This was done because of intractable pain secondary to chronic duodenal ulcer. During the next 13 years the patient was in hospital several times with the diagnosis of acute gastritis and pancreatitis.

Roentgenographic studies on several occasions over a period of four years after the operation were unremarkable. Then, in 1958, a condition that later was found to be prolapsis appeared, but it was erroneously interpreted as an extrinsic mass, and this impression continued in numerous examinations until 1967.

In 1967, the patient complained of vomiting an hour and a half after even small meals. No evidence of gastrointestinal bleeding was present. A roentgenographic diagnosis was made of prolapsed gastric mucosa associated with intermittent stomal obstruction. Comparison with the 1958 film showed the extent of prolapse had doubled in the interval. Fluoroscopic observations at the time prolapse was diagnosed in 1967 were most striking. The gastric pouch would accept about 400 ml of liquid barium. Emptying in the upright position was greatly delayed, with only small amounts of contrast material passing from the pouch into the small bowel at any one time. As the small bowel filled, the large prolapsed mass of the gastric mucosa was visible. (See Figure 1.)

The redundant gastric mucosa was excised and the histologic report described extensive hyperplasia of the gastric glandular epithelium. There was extensive edema and moderate lymphocytic and plasmocytic infiltration throughout. The final diagnosis was benign polypoid hyperplasia of the gastric mucosa.

At last report in mid-1969, the patient no longer had symptoms of gastric outlet obstruction. However he had symptoms related to "dumping," characterized by postprandial weakness and diarrhea. His weight was stable.

Case 2. A 53-year-old man had subtotal gastrectomy with gastrojejunostomy of Hofmeister type in 1963 for intractable pain associated with

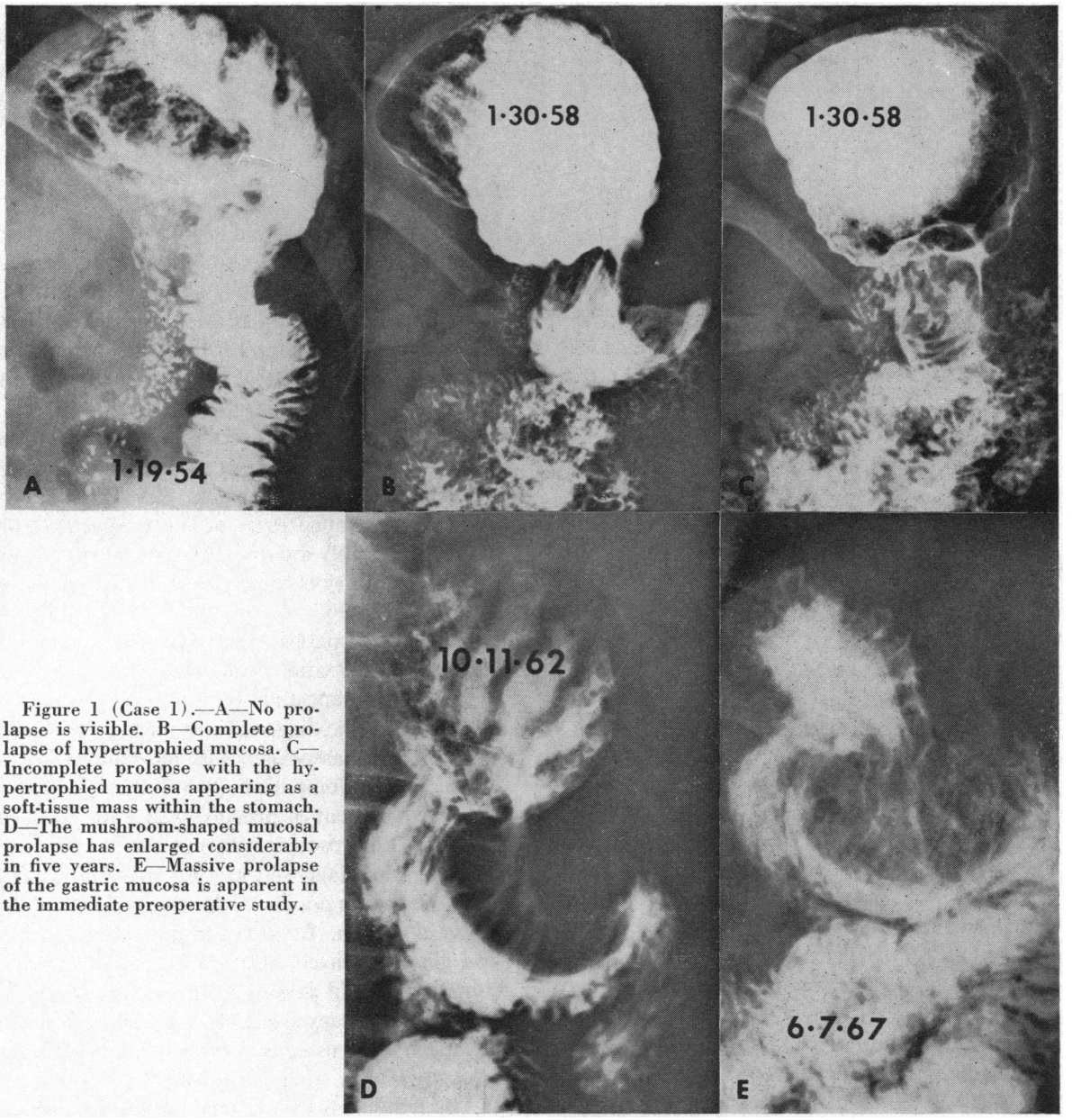

Figure 1 (Case 1).—A—No prolapse is visible. B—Complete prolapse of hypertrophied mucosa. C—Incomplete prolapse with the hypertrophied mucosa appearing as a soft-tissue mass within the stomach. D—The mushroom-shaped mucosal prolapse has enlarged considerably in five years. E—Massive prolapse of the gastric mucosa is apparent in the immediate preoperative study.

long-standing peptic ulcer. The patient had progressed well until the middle of 1967 when anorexia, nausea, weakness and dizzy spells appeared. He vomited occasionally, usually after a heavy meal. He had lost 20 pounds in the four months before admission. There was no evidence of gastrointestinal bleeding. Prolapse of gastric mucosa was diagnosed roentgenographically (Figure 2) and the redundant tissue was excised. The pathologic changes were similar to those of Case 1. After the operation the patient began to regain weight. Anorexia and weakness disappeared. When last observed, in March 1969, the patient was entirely asymptomatic.

Only a single postoperative roentgen examination was carried out following the original gastric operation. It showed the large mass at the anastomotic area to prolapse into the jejunum (Figure 2). The fluoroscopist did not observe any obstruction to the flow of barium from the stomach to the jejunum.

Discussion

In both the cases here reported, the symptoms of weight loss, nausea and vomiting after meals pointed to an obstructive process. Delayed gastric emptying, which was secondary to stomal obstruction by prolapsed mucosa, was demon-

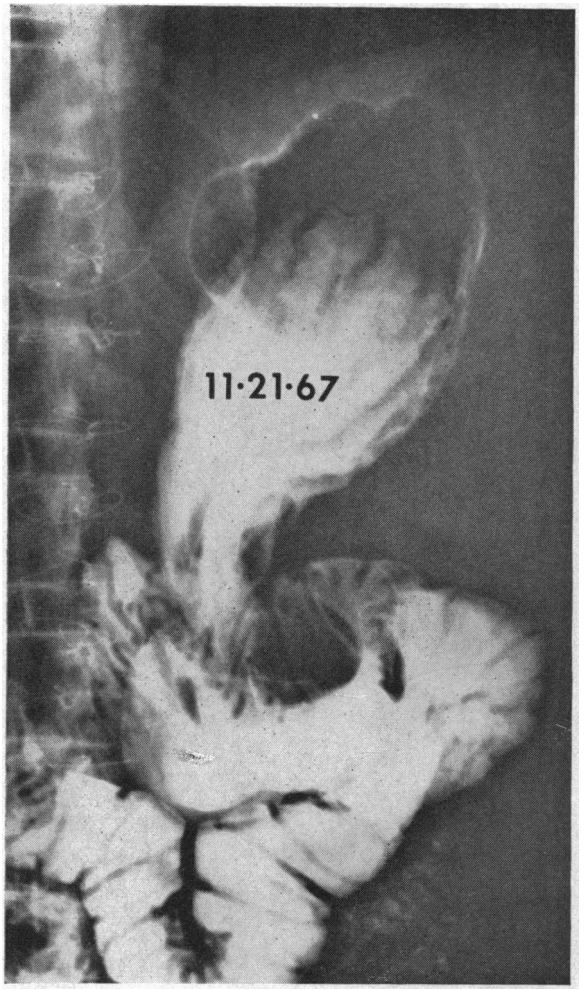

Figure 2 (Case 2).—Prolapsed gastric mucosa is seen as a mass just distal to the stoma within the jejunum.

strated fluoroscopically in Case 1. It would seem logical that obstruction would be increased, depending upon the volume and nature of the preceding meal. Surgical revision of the anastomosis with removal of the obstructive prolapsing mucosa resulted in prompt relief of symptoms in both patients.

In the series reported by LeVine and coworkers,[2] bleeding occurred in six of fourteen patients. In one case the mucosal prolapse was proved to be the site of hemorrhage.

Although the afferent loop syndrome has not yet been observed with this condition, a severe enough prolapsed mucosal mass might well cause it in some patients. We must also emphasize that milder cases of prolapse, apparently entirely asymptomatic (Figure 3), have been observed by us as well as by others.[2]

The roentgenographic appearance of prolapse of the gastrojejunal mucosa is ordinarily quite characteristic. Within the stomach, the gastric folds at the stoma may be somewhat stretched. The stoma may or may not be partially obstructed. The prolapsed mass is not clearly seen until after the jejunum fills and outlines the inferior rim. Peristaltic contractions of the jejunum alter the appearance of the mass. Peristalsis tugs on the mass in an attempt to propel it distally and then releases it, allowing the mass to retract toward the stomach. The mass may retract completely into the gastric pouch, and this possibility must be transmitted to the surgeon. In one of the cases reported herein, jejunotomy was done and no abnormality was seen until tilting of the operating table caused the redundant tissue to sag.

In our patients and in those previously illustrated, the prolapse appeared to be fairly symmetric. In distinguishing the prolapse from an extrinsic mass, all projections must show the mass to be intraluminal and surrounded by the circular folds of the jejunum.

The differential diagnosis would include the following considerations:

- Jejunogastric intussusception is the opposite situation and the mass with the characteristic jejunal folds is found in the gastric pouch.[3] When the intussusception is reduced, the jejunum appears normal.
- A more serious problem is that of neoplasm at the anastomotic junction, which would be particularly troublesome had the subtotal resection been done for gastric cancer.

While no radiologic features such as lack of distensibility or flexibility of the residual pouch were observed in the present cases, gastric cytologic examination was conducted before operation, as the surgeons were aware that a malignant tumor was a possibility. In both cases the results of cytologic studies were negative.

The only experimental evidence to support an etiologic agent is that reported by LeVine and coworkers,[2] who reproduced the roentgenographic appearance in a dog. The investigators created a gastrojejunal anastomosis by dissecting out a 2-inch mucosal cuff, which was folded back on itself as for colostomy stomas. They theorized that because of its smaller stoma the Hofmeister anastomosis was more likely to prolapse than was the Polya type. Another consideration was the occasional disparity in size between the gastric and jejunal stoma. When the diameter of the gastric

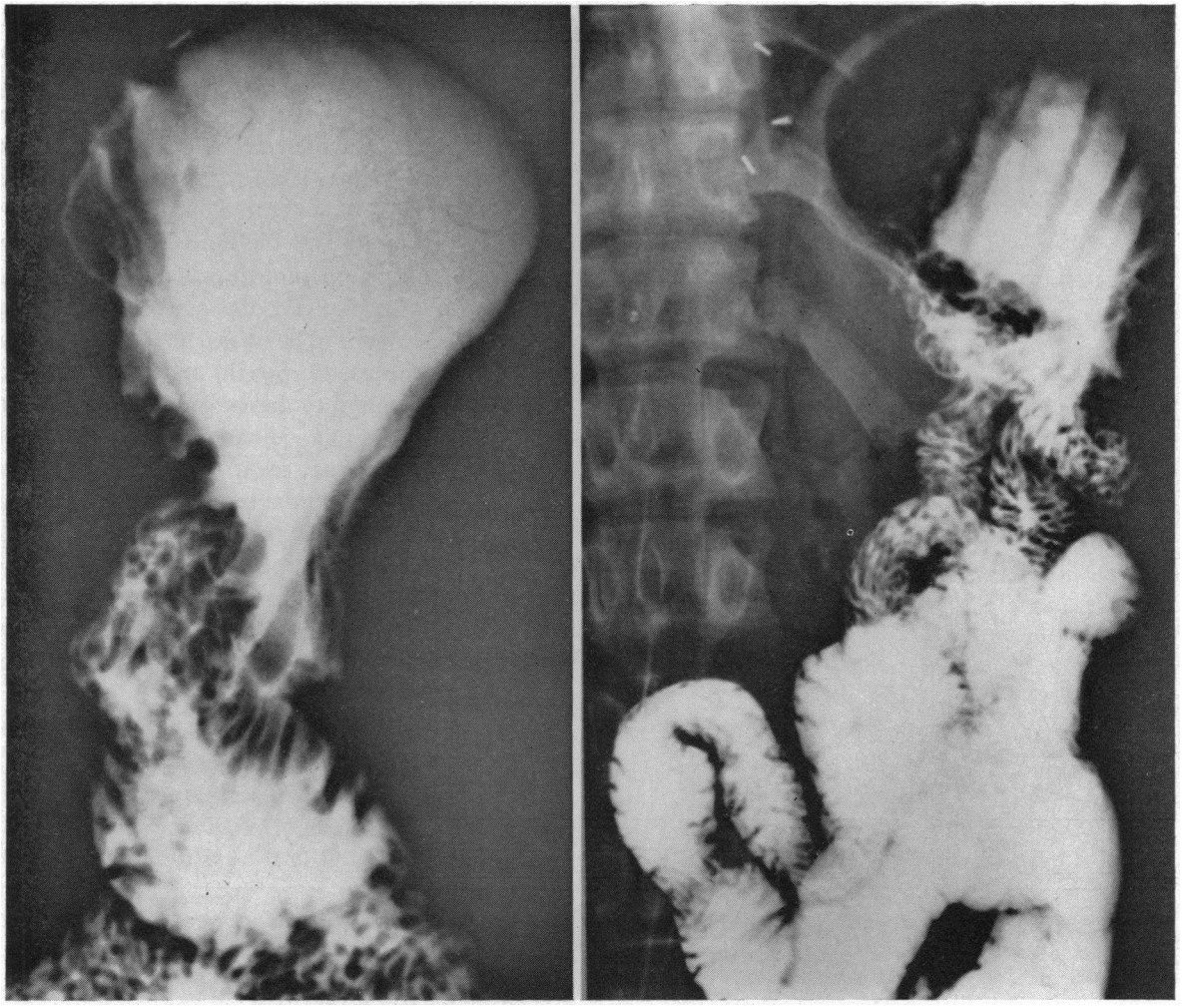

Figure 3.—*(Left)* **Demonstration of small gastric mucosal prolapse just distal to the stoma.** *(Right)* **Reduction of prolapse, with mass in the gastric pouch.**

remnant is greater than that of the jejunotomy, the piled-up gastric mucosa is pleated about the stoma, tending to prolapse subsequently.

Grimoud and coworkers[4] theorized that hypermobility of the mucosa secondary to edema of the submucosal layer and hypertrophy of the mucosal folds are necessary to generate mucosal prolapse. These changes, combined with the technical considerations posed by LeVine, may explain the pathogenesis of the gastrojejunal mucosal prolapse.

Our observations in Case 1 provide convincing evidence that, whatever the initiating factors, the process may be a progressive one. Edema is known to be a factor in the size of the mass. The proliferation of gastric mucosa, however, is assumed to be the factor that transforms a frequently asymptomatic postoperative remnant into disabling illness.

The critical elements of mucosal prolapse are the possibilities of either obstruction or hemorrhage. In the absence of these complications and with negative gastric cytologic results, periodic follow-up examinations would seem appropriate.

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